

Of the many things which have happened this past year, or any year for that matter, only the free-wheeling crystal-gazer can hope to pick out those which time will prove to be the most significant. We make no serious claim for such clairvoyance and yet the writing of an Annual Report demands that we set down what, from the present short range point of view, seem to have been major accomplishments in 1962. Accordingly we would suggest the following:

1. Dr. Grant's book, *Toxicology of the Eye*. This is the first comprehensive book on poisons of the eye since a long out-dated collation on the subject by two German authors in 1914! Dr. Grant's book, which has been more than a decade in preparation and contains many original observations in addition to extensive review of other works, is certain to be a standard of authority for years to come.

2. Dr. Kinoshita's elucidation of the pathogenesis of sugar cataracts. Although undertaken with particular reference to diabetes and galactosemia, the observations provide a model for investigation of cataractogenesis in general. This is, perhaps, the first comprehensive demonstration of the biochemical processes involved in cataract formation.

3. Dr. Friedman's technique for visualization of the retinal circulation during life. This new method of preparation opens great possibilities for study of dynamic processes at the capillary level and for correlation of microcirculation with the extensive histologic observations that have been made in this Laboratory by Dr. Kuwabara and others during the past several years.

4. Dr. Kaufman's use of 5-iodo-2'-deoxyuridine for herpetic keratitis. The discovery was noted in last year's Report and the experience of this past year has confirmed the effectiveness of this agent as a viral antibiotic. The blinding and semi-blinding devastations of herpes simplex keratitis have already been prevented in many patients by this agent. Dr. Kaufman has moved on to the chairmanship of the Department of Ophthalmology at the University of Florida but his accomplishments at the Howe Laboratory and at the Infirmary were epochal landmarks in combating ophthalmic disease.

Having listed somewhat arbitrarily these four specific accomplishments, we cannot say the other studies are less important; namely those concerning the histophysiology and biochemistry of

the retina, electron microscopy of the eye, the dynamics of intra-ocular fluid movement, instrumentation, teaching program, or the many other activities that took place in the Howe Laboratory this past year. As has been our custom these will be described under categorical headings.

VASCULAR SYSTEMS OF THE RETINA

When Dr. Kuwabara discovered three years ago that trypsin digestion and mild manipulation of the excised retina would isolate the intact retinal vasculature from its surrounding tissue while preserving the vessels sufficiently to permit use of cytologic stains, he introduced a valuable new approach to the study of small vessels in the retina. One of the first consequences was identification of a type of cell, now called mural cell, which appears to regulate flow through the complex of capillaries. These mural cells disappear characteristically in diabetes. This year we have come to realize that alteration of vascular resistance to blood flow following loss of these mural cells leads to formation of shunt vessels and that herein lies the pathogenesis of diabetic retinopathy. Not only do the shunt vessels develop the characteristic diffuse and aneurismal dilatations but the shunt results in a closing down of the bypassed vessels. Much of the vascular disease in the retinas of diabetics appears to result from this rerouting of blood.

Our present research into the pathogenesis of diabetic retinopathy is thus shifting from the morphologic study of microaneurisms to the study of the metabolic and functional activities of the mural cells. These cells are, so far as we now know, unique to the retinal vessels, and have histochemical properties that distinguish them from endothelial cells. This difference may be the key to disclosing the specific cause of the retinal vascular disease in diabetes because in diabetes the endothelial cells proliferate while the mural cells atrophy; in most other conditions (age and malnutrition) the endothelial cells disappear while the mural cells survive.

To obtain essential information on the dynamic aspects of the retinal microcirculation, it was early evident that we would need a preparation where blood-flow through the capillaries could be visualized during life. Dr. Ephraim Friedman has, during the past year, accomplished this to a degree beyond expectations. By removal of a scleral button from anesthetized cats' eyes followed by careful dissection of the choroid and outer layers of the retina,

Dr. Friedman is now able to focus on the retinal capillaries with microscopic magnifications of 1000 times. This permits visualization of individual blood corpuscles and details of the capillary walls; the observations are being documented by cinematography.

Electron microscopy is also being utilized to define the structure of the cells and membranes constituting the retinal capillary walls. In the ultrastructure of mural cells and of endothelial cells, so far, only minor differences have been discerned, insufficient to permit inferences as to differential functions. The membrane of the retinal capillary wall appears considerably thicker than that of capillaries elsewhere and shows gross alterations with age and disease. We hope to have sufficient material eventually for interpretation of these changes but electron microscopy is a laborious process and control material from human beings is available only infrequently; it will be some time before sufficient data are obtained for reliable conclusions.

Glaucoma Research has again been characterized by a broad approach ranging from experimental laboratory to clinical therapeutic investigations. This has been facilitated by close association between the Howe Laboratory and the Glaucoma Consultation Service of the Massachusetts Eye and Ear Infirmary, with most helpful cooperation from members of the ophthalmic staff of the Infirmary. Doctor Chandler has as usual provided invaluable counsel and stimulation. Research fellows, who are already graduate ophthalmologists, are given opportunities to carry on clinical investigations. This year Dr. Frank P. English from Australia and Dr. Jose D. Peczon from the Philippines have contributed in particular to studies on so-called consensual pressure change, on new types of tonometers for evaluating intraocular pressure, on the use of suction cups in experimental alteration of intraocular pressure, and on making a photographic record of changes in the angle of the anterior chamber.

Dr. Grant has supervised the activities of the Glaucoma Consultation Service and has continued systematic clinical and experimental investigations of congenital glaucoma in particular, and of the aqueous outflow channels of adult normal and glaucomatous eyes which have come to the laboratory for study (usually post-mortem). A time-consuming project of compiling in special punch-card form the vast amount of information constantly accumulating on glaucomatous patients is gradually being accomplished, with

the expectation that adequate study of the results of the past may provide valuable guidance to improvement in the future.

Specific advances to be reported are: an improvement in the treatment of malignant glaucoma, an explanation for so-called consensual pressure fall during tonography, and a simple means for making applanation tonometry easier to perform.

A group of cases of malignant glaucoma has been collected by Drs. Chandler and Grant, demonstrating successful relief of this condition by a simple form of treatment which now seems logical (utilizing cycloplegic-mydriatic drops), but was not previously appreciated or employed during the nearly one hundred years that malignant glaucoma lived up to its name as an extremely serious (though uncommon) clinical problem.

During the past 12 years an intriguing phenomenon of decrease in pressure in one eye during several minutes application of a tonometer to the other eye has been the subject of complex though inconclusive speculation and experimentation in several laboratories and clinics, but now this phenomenon is found to have relatively simple explanation. It appears to be due to evaporation of water from the eye while the lids are open and unblinking under local anesthesia. It can be prevented by covering the eye with a piece of transparent plastic film stretched from forehead to cheek and alongside the nose. Routine use of such a covering has been found to be a worthwhile adjunct and improvement in the technique of tonography.

A simple practical aid to easy performance of tonometry by means of the Goldman applanation tonometer has been provided by experimentally establishing the optimum concentration of fluorescein to be used in this procedure. Applanation tonometry is of well recognized clinical importance, but its proper performance is critically dependent upon obtaining suitable fluorescence in the tear film. This has been a troublesome variable to control by previously recommended techniques involving fluorescein-impregnated paper strips, and experience has shown this to constitute the main difficulty to be overcome in learning the use of the applanation tonometer. By experimentation it has now been established that use of a drop of a solution standardized at $\frac{1}{4}\%$ sodium fluorescein consistently and conveniently provides the appropriate fluorescence and makes applanation tonometry easier to carry out.

Biochemistry of the Lens and Cataracts. Two of the most effective means of inducing cataracts experimentally in animals are

either feeding large quantities of galactose or inducing diabetes with alloxan. It is important to understand the pathogenesis of these sugar cataracts, not only because both galactosemic and diabetic cataracts occur in human beings but because an understanding of what occurs in these experimental cataracts may give a clue to what occurs in other types of cataracts that affect mankind. This past year studies by Dr. Kinoshita, assisted by Mr. Merola and Mr. Dikmak, have gone a long way toward elucidating the underlying mechanism.

The primary changes in these experimental cataracts are swelling and hydropic degeneration of the lens fibers. The galactose enters the fibers and is reduced to its sugar alcohol, dulcitol, but in this form it cannot diffuse out of the fibers. Nor can it be metabolized. It thus accumulates to create a hypertonicity within the fiber that in turn, results in water movement into the fibers with consequent swelling, bursting and eventual disintegration of the fibers. A similar sequence of events occurs with experimentally induced diabetic cataracts. The glucose is reduced to its sugar alcohol, sorbitol, which is trapped in the fibers and exerts a similar hypertonic effect. Unlike dulcitol, however, sorbitol can be oxidized to some extent by an enzyme, polyol dehydrogenase. The hyperglycemia of diabetes is thus somewhat less effective in producing experimental cataracts than is galactosemia. Both, however, show the same biochemical and morphologic sequence of events affecting predominantly the young, and showing changes most marked in the para-equatorial portions of the lenses.

Study of the biochemical processes involved has been greatly facilitated by a technique for producing these sugar cataracts in lenses surviving in sterile culture after their removal from the eye. It has been shown by this means that the water movement into the lens parallels the accumulation of sugar alcohol and that such movement can be prevented by proportionately increasing the osmotic pressure of fluid on the outside of the lens. The very practical question is, of course, whether or not cataracts can be prevented in living animals, and ultimately in human beings, even in the presence of hypergalactosemia or hyperglycemia. Theoretically, inhibition of the enzyme, aldose reductase, which converts sugars to alcohols would prevent the development of cataracts, despite the presence of excess sugars. Present studies by Drs. Kinoshita and Hayman are directed toward factors controlling the activity of this enzyme.

Although transparent, the lens of the eye is one of the most

densely proteinaceous tissues in the body. Its proteins have long been separable into four main fractions, but the possibility has been entertained that these fractions might merely represent modifications of one or two main proteins. Dr. Spector has now analyzed the amino acids in pure samples of each of the fractions and found them to be so different that it appears unlikely they were formed by simple transformation of a parent protein.

Dr. Spector has isolated and purified an enzyme from the lens capable of removing a single amino acid at a time from the amino end of a polypeptide chain. This enzyme attacks native lens proteins only after some slight alteration has made them susceptible. Once the preliminary alteration of the lens protein occurs, the enzyme causes a rapid hydrolysis. The physiologic and chemical mechanisms which convert lens protein into a suitable substrate for this enzyme are currently being investigated. Possibly the essential initial change is similar to that which may initiate certain clinical types of cataracts induced by intoxication, inflammation, and injury.

Biochemistry of Photoreception and of the Retina. A major division of biochemical studies concerns the process of photoreception. During the bleaching of rhodopsin (visual purple), light results in a separation of retinene (vitamin A aldehyde) from the protein, opsin, and the released retinene is reduced to vitamin A. This reduction is catalyzed by an enzyme, alcohol dehydrogenase, in the visual cells, and the reducing agent has been thought to be DPNH (reduced diphosphopyridine nucleotide). Dr. Futerterman has now shown, however, that when the reduction of vitamin A aldehyde in the visual cycle is coupled to the metabolism of glucose, it is TPNH (reduced triphosphopyridine nucleotide) rather than DPNH which is the major reducing agent. The TPNH is supplied by the metabolism of glucose through the hexose monophosphate shunt pathway. The bottle-neck in the operation of this pathway is the sluggishness in the retina of mechanisms for the oxidation of TPNH to TPN. A finding which may prove to be particularly important is that alcohol dehydrogenase and the dehydrogenases of the shunt mechanism can interact so as to stimulate the oxidation of glucose by the shunt pathway. This pathway would, therefore, be expected to operate slowly in the dark and to show a burst of activity when the retina is exposed to light. In this way TPNH may automatically be manufactured somewhat in proportion to demand.

The transfer of vitamin A from the pigment epithelium to the photoreceptors and back requires esterification. Some of the factors involved in this process are being studied by Dr. Andrews and Dr. Futterman and it is hoped that in time a fuller understanding of how this is accomplished can be fitted into the picture of retinal photoreception.

Histophysiology of the Retina. One cannot work with the retina without becoming fascinated by its complex and wonderful structure and the means it provides for the conversion of light into neural impulses and for the integration of those neural impulses into meaningful messages. While Dr. Futterman is concerned, as noted previously in this Report, with the biochemistry of the photosensitive pigment, Dr. Kuwabara and Cogan have been concerned with the interrelationship of the glial (or interstitial) and neuronal portions of the retina. Müller's cells which comprise the modified glia of the retina provide the glucose requirements of the neurones and constitute the intermediate pathway between blood vessels and nerve cells. This is especially serviceable for a tissue that has no intercellular space. The observations demonstrating this functional synergism have been documented in a series of papers over the past several years and were summarized this past year in the Sanford R. Gifford Lecture by Dr. Cogan.

At present the electron microscopic characteristics of Müller's cells, and other retinal glia are being investigated most energetically. As this Report goes to press a previously undiscovered structure has been found by Dr. Kuwabara in electron micrographs of certain glia of the human retina. No further description of it will be given at present other than to point out that unanticipated discoveries of this sort make for the exciting stuff of which research is made and sometimes lead to important results.

Adding to the overall interest in glia, Dr. Simmons Lessell, a preresident Fellow, is exploring the use of silver and gold "stains" for demonstrating retinal glia. These are classical methods for studying cytology of the nervous system but they have infrequently been applied to the retina and have not previously been correlated with other histologic methods. At the same time Dr. Kuwabara is utilizing an indicator of enzyme activity, blue tetrazolium, as a histologic marker for certain glia. These combined efforts, together with the previous observations, promise to give a well-rounded understanding of the histology and function of the

retinal glia. Some of these results are to be included along with descriptions of the rest of the eye in a chapter of a forthcoming book, Greep's "Histology."

For his many contributions to ophthalmic research, and most recently to histopathology of the retina, Dr. Kuwabara was awarded this past year the New England Ophthalmological Society Annual Prize.

Toxicology of the Eye. This has been the subject of a book published this year under the authorship of Dr. Grant. This book provides in encyclopedic form a synopsis of what has been learned during the last 100 years about the undesirable effects of chemicals and drugs on the eye and on vision, either externally or systemically. It also includes the results of several years of experimentation in the Howe Laboratory on chemical injuries and their treatment.

Information on ophthalmic toxicology is scattered through many journals and books written in several languages. The intent in compiling and publishing the present compendium was to make this information more easily and readily available to physicians, chemists, industrial hygienists and others who may be confronted with practical and sometimes urgent problems in this field.

Tapetum. The tapetum lucidum is a light reflective membrane in the eyes of some nocturnal animals that is believed to enhance the visibility of these animals at low levels of illumination. Because cats and dogs which are being used for various studies in the Laboratory have these tapeta it was thought essential to have more information on the ultrastructure and chemistry of this membrane than was available in the literature.

Dr. Arnold Kroll has found with the electron microscope (and much night work, since he is combining investigation with an active residency program) that the tapetum consists of laminated cells containing dense rodlets of undetermined nature arranged in remarkably orderly fashion. One of Dr. Kroll's electron micrographs illustrating these cells was selected for the cover of Science (July 27 issue) because of both its esthetic and its scientific appeal.

Dr. James Elliott who has joined the Laboratory staff after serving his ophthalmic residency in Oklahoma, has undertaken a biochemical investigation of the tapetum lucidum. This study done in cooperation with Dr. Sidney Futterman has indicated that, in addition to remarkably high concentrations of zinc and cysteine, the cat's tapetum contains a greater concentration of riboflavin

(identified by chromatography) than has been found in any other tissue. In fact it appears to be the riboflavin which gives the tapetum its characteristic property of fluorescence.

Instrumentation. For the past four years Dr. Donaldson has been devising and perfecting a new apparatus for charting fields which has some unique features. This piece of equipment has now been completed and used satisfactorily on a number of patients. Both central and peripheral fields can be performed by the use of a large (five foot) hemisphere. The patient's fixation is monitored by a device which calls attention to loss of fixation by altering a sound signal. Fixation is governed objectively in this manner by optical and electronic means. Provision is made for automatic recording of visual field measurements and for the use of a wide range of test objects. It is felt that the equipment eliminates most of the variables in the clinical determination of visual fields by removing the various distractions inherent in most field testing devices. Moreover, it allows one to perform an accurate field determination in less than one-half the time usually required, thus decreasing the fatigue factor in the patient (and doctor).

The glass light pipe devised by Dr. Donaldson, making use of the new technology of glass fiber optics, and described in last year's Report, has now been subjected to testing and use. Unanticipated results have been obtained when it is utilized for fundus photography. When this intense light source is placed on the conjunctiva over the pars plana region, the fundus is both transilluminated and directly lighted. The appearance of the fundus stereoscopic photographs is quite different from that obtained when the fundus is illuminated through the pupil in the conventional manner. The back lighting emphasizes vessels and retinal defects not ordinarily appreciated.

Work has been continued by Dr. Donaldson on a piece of equipment to produce a pair of stereoscopic drawings by the scanning of any three-dimensional object. Particularly noteworthy has been the production of semidiagrammatic drawings of the ocular motor and visual pathways from actual anatomical preparations. Repeated changes and improvements have been required to make it possible to trace out the intricate pathways, and now it appears that, with the help of Mr. Glickman, some valuable teaching material in the field of neuro-ophthalmology can be produced.

Neuro-ophthalmology. The close rapport of the Howe Laboratory with the neurology services of the Massachusetts General

Hospital continues to provide excellent opportunity for clinical research and accomplishment. Several Fellows supported by the National Institutes of Health and by the E. B. Dunphy Fellowship (Massachusetts Lions Clubs) have served for a year each as trainees. This past year several specific studies have been undertaken including an evaluation of pain in the homolateral eye as a diagnostic sign of vascular lesions in the occipital lobe (Dr. D. Knox) and a collection of clinical material (Dr. Cogan) for a possible text on neurology of the visual system.

The projection of the retina, most especially of the macula, on the lateral geniculate body is part of a long-range project of Dr. Kupfer. Through the lateral geniculate body must pass all the impulses from the eye to the higher visual centers in the brain. The functions of this relay and especially of its curious lamination are not understood. Needed as a first step in its investigation is a determination of the topical representation of the retina in this relay. Since this requires observation on selected autopsy material, collection of data is necessarily slow.

Miscellaneous. In research devoted to exploring the unknown. many projects are tried out on a pilot basis only to be given up when found to be unfeasible or impracticable. Others may yield definite answers but are of insufficient magnitude to warrant special sections in this year's Report. Some of these projects are noted herewith.

The possibility of identifying sites of dehydrogenase activity in electron micrographs of retinas incubated with tetrazolium and suitable substrates seemed promising one year ago. The precipitated tetrazolium was electron-opaque, and preliminary observations on the retina showed it to be satisfactorily localized in the mitochondria of the retina. Further experience by Drs. Kroll and Kuwabara has shown, however, that contrary to reports in the literature much of the tetrazolium was dissolved and removed by all types of embedding media now used for electron microscopy. The method was therefore thought to be unsuitable for the dehydrogenase study and the project has not been pursued further.

One by-product of this study was, however, the important observation that the presence of ATP (adenosine triphosphate) would prevent swelling of mitochondria in tissue subjected to incubation. This finding can be utilized to facilitate the preservation of cellular ultrastructure for electron microscopy of incubated tissue.

Now that the electron microscope is in full and satisfactory operation we plan to make numerous examinations of biopsy tissue that becomes available from the operating room. So far Dr. Kroll has studied the ultrastructure of melanomas of the eye and rhabdomyosarcomas of the orbit. Dr. Kuwabara has prepared electron micrographs of various tissues of the globe for background reference, and Dr. Oikawa is preparing serial electron micrographs of the retina.

As noted in previous Reports, implantation of polyethylene tubes into the eyes of rabbits has enabled Dr. Kupfer to monitor the outflow facility of aqueous humor. Studies using this technique are being pursued in cooperation with Drs. Aaberg and Garcia. Preliminary data indicate that outflow resistance is not constant but varies with the rate of perfusion.

Several cases of keratitis resulting from exposure to a pediculocide, A-200 pyruvate, were studied by Dr. Reinecke. The toxic agent was found to be a detergent in the solution.

The process by which white blood cells reach corneal wounds was investigated by Drs. Robb and Kuwabara. Since corneas normally have no blood vessels white blood cells have an unusual problem in gaining access to central corneal abrasions. By systematic investigation of wounds of rabbit corneas it was found that the white blood cells actually penetrate the conjunctival epithelium and reach the cornea by way of the tear film. Although contrary to most modern teaching this route of the white blood cells was actually suggested by Julius Cohnheim, in 1867(!) soon after their first identification under the microscope.

An exhibit prepared by Drs. Kupfer, Donaldson and Mr. Glickman and based on the gonioscopic appearance of the angle of the anterior chamber in infants' eyes, was awarded second prize at the meeting of the American Academy of Ophthalmology and Otolaryngology this past November. This exhibit resulted from a study extending over the past several years in which the angles were photographed in the gross specimen. The eyes were then sectioned and transparent three-dimensional reconstructions made with the various components of the angle represented by different colors. Especial emphasis was placed on the spongy tissue which distinguishes the angle of the infantile eye from that of the adult. The identification of the ciliary body and scleral spur used as criteria in assessing the appearance of the adult angle cannot be used for the infant since these structures are obliterated by the spongy tissue within the angle. The most important landmark in the

infant angle is Schlemm's canal, made visible by filling it with blood. This is accomplished following the application of gentle pressure on the eye.

ORGANIZATION

Anyone who has the opportunity of sitting on both lay and scientific boards charged with administration of research must perceive the fundamentally different approach which these two groups have toward the most effective organization of research. The lay groups are comprised mostly of lawyers and business men of unusual competence; they are impressed by order and authority. The scientists, on the other hand, generally feel that new ideas are apt to derive from most unordered circumstances and sometimes from most unauthoritative sources; they are irritated by regimentation of their work.

In our attempts to maintain a reasonable compromise between order and chaos we are reminded of an analogy, attributed to Sir Charles Dodds, whereby research is likened to a man stumbling across a dark room, trying to find the light switch and upsetting the furniture as he goes. Once that he has found the switch and the light is on he puts all the furniture in order again. The operation of the Howe Laboratory also has a generous measure of stumbling and ordering.

With the devotion of the Howe Laboratory to bridging the clinical and basic sciences it was felt expedient to have a single chief for the research and clinical activities at the Massachusetts Eye and Ear Infirmary, and of the Department of Ophthalmology of the Harvard Medical School. Accordingly, on Dr. Dunphy's retirement, Dr. Cogan was appointed Chief of Ophthalmology at the Infirmary and of the Department of Ophthalmology of the Harvard Medical School. He will be assisted by Dr. Carl Johnson who was appointed Associate Chief in Charge of Clinical Services. It is our hope and aim to continue the progressive union of research and practice which developed so happily with Dr. Dunphy.

Expansion of research activities is anticipated for the near future in a new building provided by matching funds of the Infirmary and National Institutes of Health. This building will be adjacent to the present Nurses' Residence, unfortunately several blocks from the present Infirmary. While this distance is unde-

sirable, no alternative seemed practicable to solve our present urgent need of space. It is our plan at present to move our basic science divisions (that is electron microscopy, biochemistry, and some physiology) to the new quarters and maintain the primarily clinically oriented divisions in our present quarters. We are somewhat worried by the physical split which this entails but plan to institute means for maintaining the rapport which has characterized the various divisions of the Laboratory in the past.

A new venture of the past year has been the establishment of a department of medical illustration within the Howe Laboratory. We are most fortunate in obtaining Mr. Jerry Glickman and have already called heavily on his assistance for the preparation of manuscripts, teaching manuals, and exhibits.

Studies with the electron microscope have proved so productive that one machine is no longer sufficient for the many demands put upon it. This past year we developed an acute need for a smaller electron microscope to be used for scanning purposes in conjunction with the present large scope. Fortunately a friend of the Laboratory represented these needs to the Concordia Foundation and as this Report is in press we have the prospect of adding this adjunctive microscope to the Laboratory.

As in previous years, several meetings have especially involved the Laboratory or its personnel. Dr. Jin Kinoshita was again the chairman (as he has been every year since its founding) of the Ophthalmic Biochemistry Conference held in suburban Boston. Dr. Carl Kupfer was chairman of the Eastern Section of the Association for Research in Ophthalmology held this year in the Science Museum. Dr. David Donaldson again organized the clinical case presentations for the New England Ophthalmological Society meetings. Dr. Grant completed his third year on the Sensory Diseases Study Section of the National Institutes of Health, and Dr. Cogan continues as Editor-in-Chief of the Archives of Ophthalmology.

A research training program was instituted at the Howe Laboratory two years ago by Drs. Dunphy and Kupfer. It was anticipated that certain few — perhaps one in four — of the prospective residents might wish to spend a preliminary year in the Laboratory prior to the clinical period. Funds were secured from the Public Health

Service to support such a venture. To our surprise and pleasure, many more than one in four — in fact the majority of residents — have elected to take this additional year of training. In several cases research projects begun on a full-time basis, have been subsequently pursued during the period of clinical training on a part-time basis. We believe the program provides better orientation of the clinician-trainee in academic problems and will stimulate some to pursue careers in investigative ophthalmology.

The Lucien Howe Library which has a close operative affiliation with the Laboratory has continued its growth and service under the stewardship of its able librarian, Mr. Charles Snyder. The new items, patronage and circulation were all greater (including the budget) than any other year and closely geared to the research, teaching, and clinical activities of the Howe Laboratory and to the Infirmary. From the Library has also come a series of articles, mostly by Mr. Snyder, entitled "Our Ophthalmic Heritage" published monthly in the Archives of Ophthalmology.

The Howe Laboratory has again had the backing of a loyal group of friends and agencies; it is primarily for them that these Annual Reports are prepared. Our efforts would have amounted to little had it not been for the generous support we have received and which we now take pleasure in acknowledging:

For General Expenses

Individual benefactors

Anonymous
Edward W. Allen, M.D.
Mr. George Storer Baldwin
William P. Beetham, M.D.
Dr. and Mrs. Harry E. Braconier
John M. Carroll, M.D.
Paul A. Chandler, M.D.
Julian F. Chisholm, Jr., M.D.
Thomas G. Cogswell, M.D.
John A. Coyle, M.D.
Edward A. Cramton, M.D.
Thomas P. Cronin, M.D.
William F. Donoghue, M.D.
Miss Winifred J. Dornhoefer
Mrs. Eugene F. DuBois
Edwin B. Dunphy, M.D.
Charles W. Dyson, M.D.
Mahlon T. Easton, M.D.

Emilio M. Gianarelli, M.D.
Herbert Giller, M.D.
Miss H. Louise Harris
Lawrence B. Holt, M.D.
Mr. and Mrs. John N. M. Howells
Miss Doris Hubbard
Paul D. Hurley, M.D.
Carl C. Johnson, M.D.
Mr. Robert Kestnbaum
Merrill J. King, M.D.
Mrs. Grace Leeds
Mr. and Mrs. Leon Levy
Dr. and Mrs. I. Mallin
Mrs. Alfred Meyer
Miss Jean McLean Morron
Henry A. Mosher, M.D.
Bertha Offenbach, M.D.
Frank A. Perreten, M.D.
Dr. and Mrs. Marvin Posner
Marion C. Putnam, M.D.
Mrs. Laurance D. Redway
Estate of Gertrude Runkle
Benjamin Sachs, M.D.
Alfred W. Scott, M.D.
David H. Scott, M.D.
Albert E. Sloane, M.D.
Mr. Paul M. Steiner
Mr. and Mrs. Brooks Stevens
Garrett L. Sullivan, M.D.
Fritz B. Talbot, M.D.
Irvin S. Taylor, M.D.
Mr. J. M. Ulmer

Memorial Funds

Christy Cogan
Dr. and Mrs. Frederic B. Breed
Dr. and Mrs. Robert J. Brockhurst
Samuel T. Clarke, M.D.
Joseph M. Clough, M.D.
Dr. and Mrs. Stanley Cobb
Miss Ann Cogan
Edwin B. Dunphy, M.D.
Trygve Gundersen, M.D.
Dr. and Mrs. Abraham Spector

Vera Frohloff
Mr. Albert H. Frohloff
I. Frohloff
Ruth E. Grabert
Virginia J. Cossaboom
North Brookfield Grange #132

Isabella M. Gustafson
Miss Emma C. Leonard

George F. Hoysradt
Mr. George M. Olive

Mrs. John I. Kane
Mrs. Kenyon Joyce

Mildred E. Olive
Mr. George M. Olive

John E. Rines
Mrs. John E. Rines

Bausch & Lomb Incorporated

Devonshire Associates

The Gaylord Fund, Inc.

Genradco Trust

Lions Orthoptic Clinic of Western Massachusetts, Inc.

Quincy United Fund

Research to Prevent Blindness, Inc.

Max, Martha and Alfred M. Stern Fund

David M. Whitney Fund

For Specific Projects

Concordia Foundation

Electron microscopy
Glaucoma research

The Fight for Sight

A study of retinitis pigmentosa in dogs
Projection of the human retina on the lateral geniculate nucleus

Massachusetts Lions Eye Research Fund

New construction

National Science Foundation

Development of equipment for producing stereograms

Research to Prevent Blindness, Inc.

Seventh Conference on Biochemistry

Alfred P. Sloan Foundation

Basic experimental studies in glaucoma

The Charles Irwin Travelli Fund

Retinal microcirculation

U. S. Atomic Energy Commission

The carbohydrate metabolism of ocular tissues

U. S. Public Health Service

Ophthalmology Training Grants

Electron microscopy of retinal dehydrogenases

Metabolic histochemistry of the retina

Pressure regulating mechanisms in glaucoma

Research Career Development Award

Lens protein and glutathione

Lipid synthesis of non-adipose tissue

Fate of the corneal graft determined by nuclear sex

Instrumentation in the field of ophthalmology

Subjective color phenomena in normal and abnormal individuals

Photographic recording of ophthalmic disease

Development of anterior chamber angle in the human eye

Control of intraocular pressure

Retinal metabolism

Projection of the human retina on the lateral geniculate nucleus

Retinal microcirculation

Also indispensable to the successful operations of the past year have been the following secretaries, technicians, and assistants who have participated in the various activities with enthusiasm and industry: Virginia C. Ayres, Inez M. Berry, Lee Chumbley, Claude Delise, Elias Dikmak, Edna Dornhoefer, Winifred J. Dornhoefer, M. Jean Garland, Jerome Glickman, Linda Glickman, Jeanne M. Grant, Peter H. Hadfield, Setsuko Hirosawa, Carol K. Kammen, Audrey Kupfer, Elisabeth B. Lavoie, Irod Lindsay, Frances Q. Mackenzie, Linda L. McKenzie, Gloria Merola, Setsuko S. Mimura, Werner Mueller, Jr., Patricia Palmer, David G. Perkins, E. Joann Perkins, Susan Pier, Wanda Pilarski, Peter Poulimenos, Mary L. Pruyn, David C. Richardson, Kenshi Satoh, Marjorie E. Saunders, Bernard L. Schainholtz, Philip G. Schwartz, Judith G. Schwerdt, Judy Siegel, Melvin J. States, Ralph Stoll, Malcolm W. Swan, Arnold C. Vanderhoop, Thaddeus L. Wandel, Olga Wehm-Dalton, Pao-Hsi Yeh, Billie Louise G. Young.

DAVID G. COGAN, M.D.

Director

PUBLICATIONS

ANDREWS, J. S.

The lipids of arcus senilis. *Arch. Ophthalm.* 68:264-266, August 1962.

COGAN, D. G.

Corneal vascularization. *Invest. Ophthalm.* 1:253-261, April 1962.

Anatomy of lens and pathology of cataracts. *Exp. Eye Res.* 1:291-295, June 1962.

with Simmons, R. J.: Occult temporal arteritis. *Arch. Ophthalm.* 68:8-18, July 1962.

with Kuwabara, T. and Richardson, E. P., Jr.: Pathology of abiotrophic ophthalmoplegia externa. *Bull. Johns Hopkins Hosp.* 111:42-56, August 1962.

Retinal architecture and pathophysiology. The Sanford R. Gifford Lecture. *Amer. J. Ophthalm.* 54:347-363, September 1962.

with Kuwabara, T.: Pathology of cataracts in mongoloid idiocy. A new concept of the pathogenesis of cataracts of the coronary-cerulean type. *Docum. Ophthalm.* 16:73-80, 1962.

Nystagmus. Chapter in: *Strabismus, Symposium of the New Orleans Academy of Ophthalmology*, edited by G. M. Haik. St. Louis, Mosby, 1962. pp. 113-122.

Editorial: Prevention of blindness. *J.A.M.A.* 180:238-239, April 21, 1962.

Editorials in the *Archives of Ophthalmology*:

A decade of NINDB of NIH. 67:120-122, February 1962.

Treatment of herpetic keratitis. 67:122, February 1962.

Triparanol and medical reporting. 67:397-398, April 1962.

Aerospace problems. 67:546, May 1962.

AFIP and ophthalmology. 67:546-548, May 1962.

Chromatin, chromosomes, and ophthalmology. 67:697-700, June 1962.

Entre aveugles. 68:1-2, July 1962.

Conference on diabetic retinopathy. 68:2-3, July 1962.

Cardiac glycosides and glaucoma. 68:155-156, August 1962.

Lasers. 68:156-157, August 1962.

Taking the malignancy out of malignant glaucoma. 68:301, September 1962.

Lens symposium at Oxford. 68:302-303, September 1962.

Therapeutic hypopituitarism for diabetic retinopathy. 68:579-580, November 1962.

DONALDSON, D. D.

A camera stand for clinical photography. *Arch. Ophthalm.* 68:518-520, October 1962.

FUTTERMAN, S.

Enzymatic oxidation of vitamin A aldehyde to vitamin A acid. *J. Biol. Chem.* 237:677-680, March 1962.

with Richardson, D. C.: Vitamin A aldehyde oxidation in the retina. *Fed. Proc.* 21 (No. 2) March-April 1962, p. 473.

- GOLDSTEIN, J. E.
with Cogan, D. G.: Sclerocornea and associated congenital anomalies. Arch. Ophthal. 67:761-768, June 1962.
- GRANT, W. M.
Toxicology of the Eye. C. C. Thomas, Publisher, Springfield, Ill., 1962, 580 p.
with Adams, S. T. and Smith, T. R.: Congenital glaucoma (possibly Lowe's syndrome). Arch. Ophthal. 68:191-195, August 1962.
with Chandler, P. A.: Mydriatic-cycloplegic treatment in malignant glaucoma. Arch. Ophthal. 68:353-359, September 1962.
- HOWARD, G. M.
with Kaufman, J. E.: Special review: Herpes simplex keratitis. Arch. Ophthal. 67:373-387, March 1962.
- HUTCHINSON, B. T.
with Kuwabara, T.: Phosphorylase and uridine diphosphoglucose synthetase in the retina. Arch. Ophthal. 68:538-545, October 1962.
- KAUFMAN, H. E.
Editorial: Interferon. Arch. Ophthal. 67:396-397, April 1962.
with Nesburn, A. B. and Maloney, E. D.: IDU therapy of herpes simplex. Arch. Ophthal. 67:583-591, May 1962.
with Martola, E. L. and Dohlman, C. H.: Use of 5-Iodo 2'-deoxyurine (IDU) in treatment of herpes simplex keratitis. Arch. Ophthal. 68:235-239, August 1962.
with Howard, G. M.: Therapy of experimental herpes simplex keratitis. Invest. Ophthal. 1:561-564, August 1962.
with Maloney, E. D.: Antibody tests for ocular toxoplasmosis. Invest. Ophthal. 1:556-560, August 1962.
- KINOSHITA, J. H.
The effect of aging on the biochemistry of the lens. Biological aspects of aging. Edited by Dr. Nathan W. Shock, Columbia University Press, New York, 1962, 305 p.
Some aspects of the glucose metabolism of the cornea. Invest. Ophthal. 1:178-186, April 1962.
with Merola, L. O. and Dikmak, E.: Osmotic changes in experimental galactose cataracts. Exp. Eye Res. 1:405-410, June 1962.
with Merola, L. O., Satoh, K., and Dikmak, E.: Osmotic changes caused by the accumulation of dulcitol in the lenses of rats fed with galactose. Nature 194:1085-1087, June 1962.
with Merola, L. O. and Dikmak, E.: The accumulation of dulcitol and water in rabbit lens incubated with galactose. Biochem. Biophys. Acta 62:176-178, 1962.
Annual Review: Physiological chemistry of the eye. A.M.A. Arch. Ophthal. 68:554-571, October 1962.
- KNOX, D. L.
with Cogan, D. G.: Eye pain and homonymous hemianopia. A.J.O. 54:1091-1093, December 1962.

KROLL, A. J.

with Kuwabara, T.: Photograph: Tapetum of the cat's eye. Cover of: Science 137 (# 3526) July 27, 1962.

Prevention of phosphate-induced mitochondrial swelling. J. Cell. Biol. 15:29-35, October 1962.

KUPFER, C.

Trans-synaptic atrophy in the human lateral geniculate nucleus following unilateral enucleation. Fed. Proc. 21, March-April 1962. (Abstract 0.354.)

Studies of intraocular pressure. II. The histopathology of experimentally increased intraocular pressure in the rabbit. Invest. Ophthalm. 1:474-479, August 1962.

The projection of the macula in the lateral geniculate nucleus of man. Amer. J. Ophthalm. 54:4, 597-609, October 1962.

with Brubaker, R.: Microcryoscopic determination of the osmolality of interstitial fluid in the living rabbit cornea. Invest. Ophthalm. 1:653-660, October 1962.

Relationship of ciliary body meridional muscle and corneoscleral trabecular meshwork. Arch. Ophthalm. 68:818-822, December 1962.

Editorial: The split-brain. Arch. Ophthalm. 68:719-720, December 1962.

KUWABARA, T.

with Cogan, D. G.: Retinal glycogen. Trans. Amer. Ophthalm. Soc. (1961) 59:106-110, 1962.

with Richlin, J. J.: Amyloid disease of the eyelid and conjunctiva. Arch. Ophthalm. 67:138-142, February 1962.

REINECKE, R. D.

with Kuwabara, T., Cogan, D. G. and Weis, D. R.: Retinal vascular patterns. V. Experimental ischemia of the cat eyes. Arch. Ophthalm. 67:470-475, April 1962.

with Kinder, R. S. L.: Corneal toxicity of the pediculocide A-200 pyriate. Arch. Ophthalm. 68:36, July 1962.

Malingering in children. Internat. Ophthalm. Clinics 2:837-842, December 1962.

ROBB, R. M.

with Kuwabara, T.: Corneal wound healing. I. The movement of polymorphonuclear leukocytes into corneal wounds. Arch. Ophthalm. 68:632-642, November 1962.

SNYDER, C.

Our Ophthalmic Heritage:

Edward Jackson. Arch. Ophthalm. 67:101-102, January 1962.

Julian John Chisolm. Arch. Ophthalm. 67:262-264, February 1962.

Charles Henry May. Arch. Ophthalm. 67:388-389, March 1962.

Silas Weir Mitchell and research in ophthalmology. Arch. Ophthalm. 67:528-530, April 1962.

The eyes of John Dalton. Arch. Ophthalm. 67:671-673, May 1962.

Von Graefe as viewed by a contemporary. *Arch. Ophthal.* 67:827-829, June 1962.

Allvar Gullstrand, Nobel Laureate. *Arch. Ophthal.* 68:139-141, July 1962.

Herman Snellen and V-d/D. *Arch. Ophthal.* 68:571-573, October 1962.

Julius Homberger, M.D. *Arch. Ophthal.* 68:875-878, December 1962.

Bates, Huxley, and Myself — A saga in visual re-education. *Internat. Ophthal. Clinics.* 2:921-934, December 1962.

SPECTOR, A.

A study of peptidase and esterase activity in calf lens. *Exp. Eye Res.* 1:330-335, June 1962.

TOUSSAINT, D.

with Cogan, D. G. and Kuwabara, T.: Extravascular lesions of diabetic retinopathy. *Arch. Ophthal.* 67:42-47, January 1962.

LECTURES

ANDREWS, J. S.

Lipid metabolism of cornea. Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts, October 16, 1962.

COGAN, D. G.

Ophthalmology, Harvard Medical School, First year class, Massachusetts General Hospital, in Boston, Massachusetts, January 20, 1962.

Retinal architecture and pathophysiology: The Sanford R. Gifford Lecture. Chicago Ophthalmological Society, in Chicago, Illinois, February 16 and 17, 1962.

Internuclear ophthalmoplegia. Neurological Institute, Presbyterian Hospital, in New York, New York, February 28, 1962.

Microscopic structure of the retina. New England Ophthalmological Society, in Boston, Massachusetts, April 17, 1962.

Research in seeing. Lions Club, in Springfield, Massachusetts, April 26, 1962.

Conference on diabetic retinopathy. National Committee for Research on Ophthalmology and Blindness, in Chicago, Illinois, May 6, 7, 8, 1962.

Anatomy of lens and pathology of cataracts. Symposium on Lens Metabolism in Relation to Cataract. Oxford University, in Oxford, England, May 21-26, 1962.

Seminar in Ophthalmology, University of Florida, in Gainesville, Florida, September 20-23, 1962.

The retinal vasculature.

Congenital oculomotor apraxia.

Ocular manifestations of the endocrinopathies. American College of Surgeons, Southeastern Pennsylvania Chapter, in Reading, Pennsylvania, September 26, 1962.

The remarkable radial fibers (Müller cells) of the retina. Wilmer Institute, Johns Hopkins Hospital, in Baltimore, Maryland, October 19, 1962.

Moderator. International Symposium on Ocular Tumors. Dedication of Institute of Ophthalmology, Baylor University College of Medicine, in Houston, Texas, November 12-14, 1962.

Participant. National Institutes of Health Conference on Training Ophthalmic Investigators, in Harriman, New York, October 25-28, 1962.

Retinal vascular disease. Postgraduate Course in Cardiology, Massachusetts General Hospital, in Boston, Massachusetts, December 7, 1962.

Ophthalmic pathology. Harvard Medical School, Second year class, in Boston, Massachusetts, December 22, 1962.

House Officer Lecture, Massachusetts Eye and Ear Infirmary:

Histopathology of cataracts, in Boston, Massachusetts, July 26, 1962.

DONALDSON, D. D.

Postgraduate Course in Ophthalmology, Harvard Medical School:

Corneal dystrophy, January 15, 1962.

Diseases of chamber angle, January 16, 1962.

Systemic diseases of anterior segments, January 19, 1962.

Neuro-ophthalmic anatomy, September 25–October 12, 1962.

Relationship of dermatological conditions to the eye. Dermatology Department, Boston City Hospital, in Boston, Massachusetts, February 2, 1962.

Gill Memorial Eye, Ear and Throat Hospital, April 2–6, 1962.

Corneal dystrophies.

Tumors and cysts of iris and ciliary body.

Differential diagnosis of lesions of the cornea.

Lesions of the angle of the anterior chamber.

Clinical examination of the eye as an aid to the diagnosis. Postgraduate Course in Pediatrics, Massachusetts General Hospital, in Boston Massachusetts, June 14, 1962.

External disease of the eye. Series of lectures to the Lancaster Courses in Ophthalmology, in Waterville, Maine, July 20–23, 1962.

Ocular photography as a means to diagnosis and treatment. Lions Club, in Belmont, Massachusetts, September 27, 1962.

Squints. Pediatric Service, Massachusetts General Hospital, in Boston, Massachusetts, December 1, 1962.

House Officer Lectures, Massachusetts Eye and Ear Infirmary:

Fundus lesions, March 27, 1962.

Iris tumors, June 21, 1962.

Corneal dystrophies, July 17, 1962.

Cataracts, December 11 and 13, 1962.

DUSHAY, F.

Discussion: Incidence of cataracts in rheumatoid arthritis with and without steroid therapy, by D. E. Holdworth, A. F. Calnan and T. B. Bayles. New England Rheumatism Society, in Boston, Massachusetts, May 18, 1962.

FRIEDMAN, E.

Choroidal vascular patterns. Alumni Association Meeting of the Massachusetts Eye and Ear Infirmary, in Boston, Massachusetts, April 16, 1962.

FUTTERMAN, S.

The enzymatic oxidation of vitamin A aldehyde. Seventh Conference on Ophthalmic Biochemistry, in Dedham, Massachusetts, February 24–25, 1962.

Chemistry and physiology of retina. Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts, October 23, 1962.

GRANT, W. M.

House Officer Lectures, Massachusetts Eye and Ear Infirmary:
Glaucoma in infants and children. February 27 and December 27, 1962.

with Chandler, P. A.: Mydriatic-cycloplegic treatment in malignant glaucoma. New England Ophthalmological Society, in Boston, Massachusetts, April 17, 1962.

Toxicology, tonometry and tonography. Series of lectures to the Lancaster Courses in Ophthalmology, in Waterville, Maine, August 23 and 24, 1962.

Participant. National Institutes of Health Glaucoma Conference, in Del Monte, California, September 26-29, 1962.

Departments of Ophthalmology and Continuing Education in Medicine and Health Sciences, University of California School of Medicine, in San Francisco, California.

Tonography, October 1, 1962.

Pharmacology of pressure-reducing drugs, October 2, 1962.

Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts.

Ophthalmic toxicology, November 2 and 7, 1962.

Pathology of glaucoma, December 14, 1962.

HUTCHINSON, T. B.

Histochemistry of retinal glycogen. Alumni Association Meeting of the Massachusetts Eye and Ear Infirmary, in Boston, Massachusetts, April 16, 1962.

KAUFMAN, H. E.

with Martola, E.-L. M. and Dohlman, C. H.: Treatment of herpes simplex keratitis with IDU. New England Ophthalmological Society, in Boston, Massachusetts, February 21, 1962.

KINOSHITA, J. H.

Chairman. Seventh Conference on Ophthalmic Biochemistry, in Dedham, Massachusetts, February 24-26, 1962.

Lens and cataracts. Department of Otolaryngology, Harvard Medical School, in Boston, Massachusetts, March 8, 1962.

Galactose cataract. New England Ophthalmological Society, in Boston, Massachusetts, March 21, 1962.

Sugar cataracts. Symposium on Lens Metabolism in Relation to Cataract. Oxford University, in Oxford, England, May 21-26, 1962.

Cataracts. House Officer Lecture, Massachusetts Eye and Ear Infirmary, in Boston, Massachusetts, July 31, 1962.

Biochemistry of the cornea and lens. Series of lectures to the Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts, October 11-26, 1962.

Metabolism of sugar alcohols in lens. Seminar of the Department of Biological Chemistry, University of Rochester Medical School, in Rochester, New York, October 29, 1962.

Discussion: Diabetic cataract by Berman, S. Association for Research in Ophthalmology, University of Michigan, in Ann Arbor, Michigan, December 3, 1962.

KNOX, D. L.

Interstitial keratitis. House Officer Lecture, Massachusetts Eye and Ear Infirmary, in Boston, Massachusetts, February 6, 1962.

KROLL, A. J.

Mitochondrial swelling and retinal histochemistry. Armed Forces Institute of Pathology, in Washington, D. C., April 11, 1962.

Electron microscopy of the retina. Department of Ophthalmology, University of Miami, in Miami, Florida, May 21, 1962.

KUPFER, C.

The human lateral geniculate body. Neuro-ophthalmological Conference, Massachusetts General Hospital, in Boston, Massachusetts, January 25, 1962.

Retino-geniculate relationship. Physiology Seminar, Stanley Cobb Laboratories for Psychiatric Research, Massachusetts General Hospital, in Boston, Massachusetts, March 7, 1962.

The relationship of the longitudinal muscle of the ciliary body and the corneoscleral meshwork. New England Ophthalmological Society, in Boston, Massachusetts, March 21, 1962.

The histopathology of experimentally increased intraocular pressure in the rabbit. Wilmer Meeting, The Johns Hopkins Hospital, in Baltimore, Maryland, April 12, 1962.

Trans-synaptic atrophy in the human lateral geniculate nucleus following unilateral enucleation. Federation of American Societies for Experimental Biology, in Atlantic City, New Jersey, April 18, 1962.

The histopathology of experimentally increased intraocular pressure in the rabbit. Association for Research in Ophthalmology, in Chicago, Illinois, June 28, 1962.

Outflow facility in the ambient rabbit. Glaucoma Conference, in Monterey, California, September 26-29, 1962.

The projection of the macula in the lateral geniculate nucleus of man. Festschrift for Professor Frank Walsh, The Johns Hopkins Hospital, in Baltimore, Maryland, October 19-20, 1962.

Aqueous humor dynamics. Lectures to the Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts, October 31-November 1, 1962.

Experimental glaucoma. Symposium on Current Research in Glaucoma. National Society for the Prevention of Blindness, in Las Vegas, Nevada, November 3, 1962.

Electrophysiology. Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts, December 6, 1962.

KUWABARA, T.

Vascular pathology of the retina. Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts, January 11, 1962.

The retina: Structural and metabolic study. Research Seminar, Otolaryngology Department, Harvard Medical School, in Boston, Massachusetts, January 25, 1962.

The retinal vessel: Its development and diabetic change. Department of Pathology, Harvard Medical School, in Boston, Massachusetts, February 27, 1962.

Neovasculogenesis in the retina. Ophthalmic Pathology Meeting, Armed Forces Institute of Pathology, in Washington, D. C., April 11, 1962.

Ultrastructure of the retina. New England Ophthalmological Society, in Boston, Massachusetts, April 17, 1962.

Diabetic retinopathy. Conference on Microcirculation and Diabetic Retinopathy. (National Committee for Research in Ophthalmology and Blindness) in Chicago, Illinois, May 7, 1962.

Changes in diabetic retinas. New England Diabetes Association, in Boston, Massachusetts, October 15, 1962.

Histochemistry of the retina, optic nerve, and cornea. Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts, October 19, 1962.

Retinal vessels. Faculty of Medicine, Kyushu University, in Fukuoka, Japan, December 19, 1962.

ROBB, R. M.

Corneal wound healing. Alumni Association Meeting of the Massachusetts Eye and Ear Infirmary, in Boston, Massachusetts, April 16, 1962.

SNYDER, C.

The Infirmary, 1861-1961, A comparison of statistics. Ladies Visiting Committee of the Massachusetts Eye and Ear Infirmary, in Boston, Massachusetts, January 10, 1962.

The effective use of ear, nose and throat literature. Otolaryngological Residents of the Massachusetts Eye and Ear Infirmary, in Boston, Massachusetts, April 27, 1962.

Sketches from the history of ophthalmology. Residents in Ophthalmology of the Massachusetts Eye and Ear Infirmary, in Boston, Massachusetts, June 26, 1962.

Bates, Huxley, and myself: A saga in visual re-education. New England Ophthalmological Society, in Boston, Massachusetts, November 21, 1962.

SPECTOR, A.

Lens peptidase and esterase activity. Seventh Conference on Ophthalmic Biochemistry, in Dedham, Massachusetts, February 25-26, 1962.

Lens aminopeptidase. Marine Biological Laboratory, in Woods Hole, Massachusetts, August 7, 1962.

Lens proteins. Postgraduate Course in Ophthalmology, Harvard Medical School, in Boston, Massachusetts, October 24, 1962. .

EXHIBIT

Kupfer, C. and Donaldson, D. D.: The gonioscopic appearance of the human angle from birth to one year. American Academy of Ophthalmology and Otolaryngology, in Las Vegas, Nevada, November 4-9, 1962.

FORM OF BEQUEST

The Howe Laboratory of Ophthalmology is an independent department of the Harvard Medical School and is jointly supported by a restricted endowment of Harvard University and by the Massachusetts Eye and Ear Infirmary.

For the information of those who may wish to contribute to this Laboratory, a form of bequest is here set forth:

I GIVE AND BEQUEATH TO THE HOWE LABORATORY OF
OPHTHALMOLOGY DOLLARS
TO BE APPLIED TO THE USES OF SAID LABORATORY.

Such bequests are managed by the Treasurer's Office of Harvard University, and the income is accredited to the Laboratory.

